# THE HUMAN TRACHEA IN VIBROACOUSTIC DISEASE

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**Introduction** Vibroacoustic disease (VAD) is caused by long-term (years) exposure to low frequency noise (LFN) (≤500 Hz, including infrasound) [1]. Pericardial thickening in the absence of an inflammatory process and with no diastolic dysfunction is the hallmark of VAD [2]. In animal models exposed to occupationally-simulated LFN, respiratory tract epithelial cell populations, particularly cila, secretory cells and brush cells, were shown to be a targed for this agent of disease [3]. In this report, tracheal fragments of two VAD patients were studied through light and electron microscopy.

**Methods** *Case 1*: 58 year-old female Caucasian, non-smoker, retired military parachutest, with 2000 logged flight hours in helicopters. Exhibits persistent cough and rhinitis, and positive C-ANCA. *Case 2*: 62 year-old retired male Caucasian, smoker, aircraft technician for 30 years, asymtomatic until diagnosed with a squamous cell lung carcinoma located in the upper right lobe. Both cases exhibit pericardial thickening with no inflammatory process and no diastolic dysfunction. Tracheal fragments were removed by endoscopic biopsy. *Microscopy*. Specimens for light microscopy were formalin-fixed, paraffin-embedded, hematoxylin, eosin and fuchsin-rhesorcin stained. For transmission electron microscopy (TEM), tracheal sections were fixed at room temperature in an aldehyde mixture, washed in buffer, postfixed in ferricyanide-reduced osmium solution, dehydrated through graded ethanol series, and embedded in Epon. Samples were sectioned in an LKB ultramicrotome, stained with uranyl acetate and lead citrate, and viewed with a JEOL 100C electron microscope.

**Results** Despite that fact that one is a smoker and the other isn't, both cases discolosed very similar images. With light microscopy, all images presented scattered areas with damaged cilia. In both cases, displastic foci were identified as well as basal hyperplasia (Fig. 1).

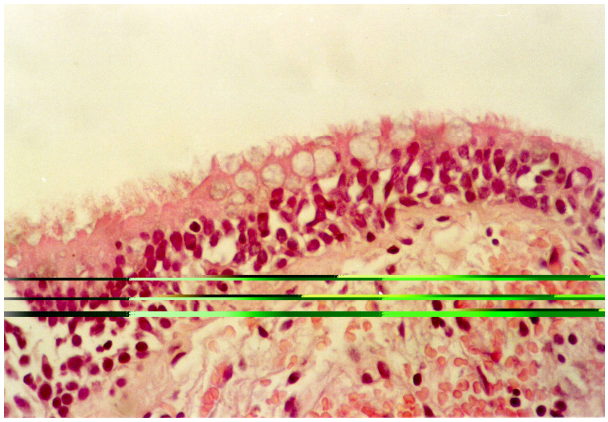

Fig. 1. Light microscpy. Tracheal epithelium of the female VAD patient, non-smoker. Basal hyperplasia and partial destruction of cilia are visible. (x 200)

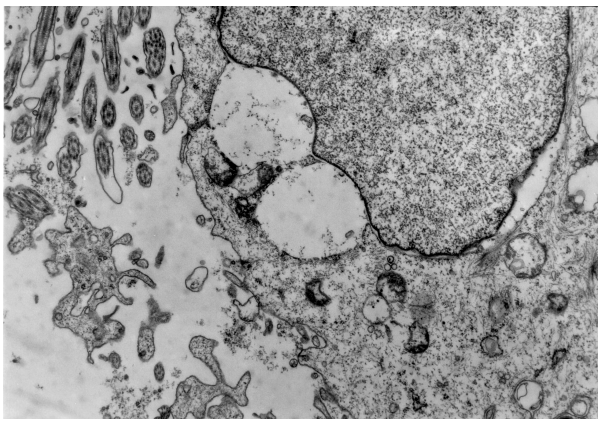

Fig. 2. TEM. Tracheal epithelium of non-smoker female VAD patient. An apoptotic cell and abnormal cilia with more than one axoneme per membrane. (x16000)

No inflammatory cellularity was identifed. In TEM, the most noticeable feature of gross changes in ciliary morphology are the multiple ciliary axonemes surrounded by a common membrane (Fig.2). The cell life-cycle seems to be greatly accelerated given the frequency of apoptotic images in all epithelial cell ultramicrographs. All cell structures seem to be under the same process of death (Figs.2-4), in contrast with a marked reinforcement of the cytoskeleton and intercellular junctions.

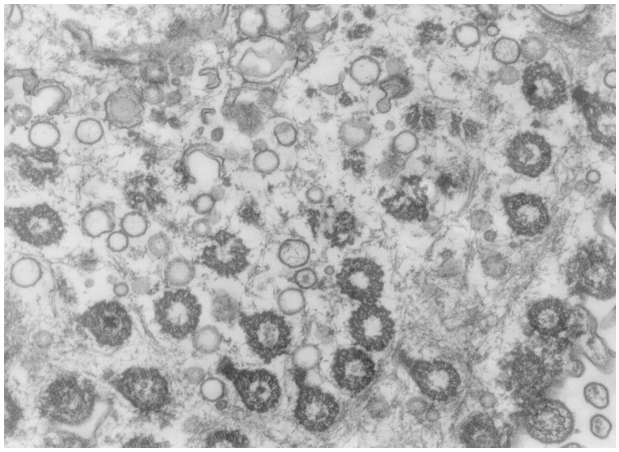

Fig. 3. TEM - Tracheal epithelium of non-smoker female VAD patient. Ciliated cell under apoptosis. In this tangential cut, the cell death process is seen to reach the basal bodies. (x20000)

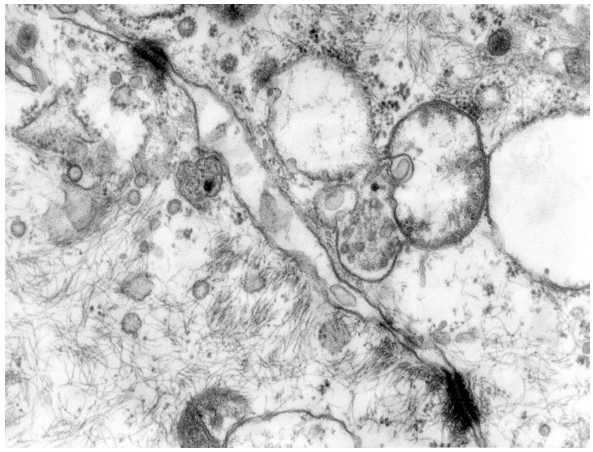

Fig. 4. Tracheal epithelium of a male VAD patient, smoker. Intercellular junctions of two apoptotic epithelial cells with impressive desmosomes and a marked reinforcement of the cytoskeleton. (x40000)

**Discussion** The cellular pattern of the observed lesions are the same in both these cases, even though one is a smoker and the other is not. Despite the fact that both patients are retired, the abnormal cellular processes seem to continue in full force, even after the LFN stessor has ceased to be present. This is in agreement with what was observed in the tracheal epithelia of rats exposed to LFN, and then kept in silence for one year, where lesions were still very visible and did not seem to be reversible [3]. The respiratory compliants yield no clues no LFN-induced pathology because only the non-smoker female has any type of respiratory symptoms: non-productive cough. Previous pulmonary functional tests were normal in most VAD patients, although high-resolution CT scan disclosed air-trapping and focal fibrosis in non-smoker LFN-exposed workers, with and without symptoms [4]. The results herein, taken together with older studies [5, for example], unequivocally link LFN to the onset of pulmonary pathology.

**Keywords:** low frequency noise, noise exposure, cilia, apoptosis, epithelial cells, pathology

**References**

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